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IDENTIFYING PREGNANT WOMEN EXPERIENCING DOMESTIC VIOLENCE IN THE EMERGENCY DEPARTMENT. *D B Nelson, E Datner, C Brensinger, D Weibe (University of Pennsylvania, Philadelphia, PA 19104)

The efficacy and cost-effectiveness of universal screening of pregnant women for Domestic Violence (DV) is unclear. Targeted strategies to identify victims of violence through determining characteristics predictive of DV are needed. DV is an extremely common public health problem and the ED represents a primary point of contact for pregnant women. This study describes demographic, socioeconomic and health status characteristics of pregnant women presenting to the ED. 1,174 pregnant women presenting to the ED at the Hospital of the University of Pennsylvania between January 1999 and August 2001 were enrolled. Information on current and past violence, sociodemographic factors, sexually transmitted diseases (STD's), depressive symptoms, and drug use were collected. Pregnant women reporting current violence were compared to pregnant women not reporting current violence. Bivariate analyses were conducted, statistical significance assessed using chi-squared and t-tests, and logistic regression models used to determine independent predictors of violence. Almost 80% of pregnant women reported prior violence and 15% of pregnant women reported current violence. Women reporting current violence were three times more likely to report depressive symptoms (OR = 3.43, 95% CI: 2.2, 5.19), over 60% more likely to use marijuana (OR = 1.68, 95% CI: 1.07, 2.63) or have a prior diagnosis of trichomonas (OR = 1.77, 95% CI: 1.15, 2.72) compared to pregnant women not reporting current violence. These results suggest that prior trichomonas, current depressive symptoms, and marijuana use are important characteristics that may be used to identify pregnant ED patients experiencing DV and to develop targeted DV screening and intervention programs.

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DIABETES TREATMENT OUTCOME IN OLDER INDIVIDUALS WITH DIABETES AND EVIDENCE OF ISLET CELL AUTOIMMUNITY. *E Barinas-Mitchell, L H Kuller, S Pietropaolo, Y-J Zhang, T Henderson, and M Pietropaolo (University of Pittsburgh, Pittsburgh, PA, 15261)

Autoantibodies (AA) to the 65 kD isoform of glutamic acid decarboxylase (GAD65), a determinant of risk for autoimmune diabetes, have been found in up to 10% of older patients with Type 2 diabetes. Using baseline stored sera from the Cardiovascular Health Study, we evaluated the relationship between baseline GAD65 AA and glucose tolerance status and current and future diabetes treatment in participants ≥65 years old (n = 3,232). The prevalence of GAD65 AA in individuals classified as normal, with impaired fasting glucose (IFG), and diabetes was 2.1%, 2.1% and 4.1%, respectively, in Whites (n = 2,729; p = 0.02) and 3.8%, 1.9% and 6.6%, respectively, in Blacks (n = 503; p = 0.13). Among participants with diagnosed diabetes at baseline (n = 666), GAD65 AA were found in 2.4%, 6.0%, 9.7% and 11.1% of participants reporting baseline use of no diabetes medication, oral hypoglycemic agents (OHGA), insulin only, and both OHGA and insulin, respectively (p = 0.006, linear trend). A similar trend was found by race and gender. Excluding baseline insulin users, insulin use at follow-up (up to 9 years) was higher in GAD65 AA positive versus (vs) GAD65 AA negative individuals among individuals classified at baseline with normal-glucose tolerance (2.9% vs 0.1%), IFG (7.7% vs 1.3%), new diabetes (14.3% vs 6.2%) and prevalent diabetes (36.4% vs 24.2%). These data suggest that even among older individuals GAD65 AA may be associated with an autoimmune-mediated insulin secretory defect and may serve as a marker of future insulin requirement.

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HOW MANY GENES DOES IT TAKE TO MAKE AN APPRECIABLE POPULATION ATTRIBUTABLE FRACTION OF A COMMON DISEASE? *Q Yang, M J Khoury, J M Friedman, W D Flanders (Centers for Disease Control and Prevention, Atlanta, GA 30333)

Most common human diseases result from complex interactions between genetic variants at numerous loci and multiple environmental risk factors. This poses a major challenge for finding and assessing the role of genes in common diseases. Although most genetic susceptibility variants probably only have a weak effect when considered individually, a combination of many such effects might account for a large proportion of disease in the population. In this study, we used the number of disease susceptibility genes and various genotype prevalences and risk ratios to calculate the population attributable fraction (PAF) for a common complex disease, assuming either additive or multiplicative gene-gene interactions. For rare genotypes (e.g., prevalence = 0.02%), many interacting genes (N >180) are needed to explain 50% of the disease in the population, even if the individual genotype risk ratios are large (RR = 10–20). On the other hand, only a limited number ($N \geq 15$) of common (prevalence = 10%) genetic susceptibility variants is needed to explain 50% of the disease in the population, even if the individual risk ratios are only weak to modest (RR = 1.5–2.0). Fewer genes are needed to produce the same PAF if gene-gene interactions are multiplicative. These results suggest that a limited number of common genetic susceptibility variants could explain a substantial proportion of common complex disease in the population. Our findings have important implications for the design and analysis of genetic studies for complex human diseases.

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RADON, GLUTATHIONE-S-TRANSFERASE M1 AND LUNG CANCER IN US WOMEN. *M R Bonner, W Xiong, W P Bennett, Q Lan, M E Wright, J H Lubin, R. W Field, M C R Alavanja (Division of Cancer Epidemiology and Genetics, NCI, NIH, DHHS, Bethesda, MD 20892–7240)

There is experimental evidence that α-radiation generates reactive oxygen species and induces oxidative stress in cells that may potentiate carcinogenesis. Glutathione-s-transferase M1 (*GSTM1*) is important in quenching reactive oxygen species and their reactive intermediates. In this study, we investigate whether there is a gene-environment interaction between exposure to α-particles from residential radon (Rn-222) exposure and *GSTM1* that affects lung cancer risk. A case-only approach was used with lung cancer cases pooled from three previously completed case-control studies. Archival tissue samples from 271 lung cancer cases were used for germline analysis to determine *GSTM1* genotype. Radon concentrations were measured with long-term alpha-track radon detectors for the period from 5–25 years prior to diagnosis. Time-weighted average of residential Rn-222 exposure was calculated for each case. Unconditional logistic regression was used to calculate the interaction odds ratios (OR) and 95% confidence intervals (95% CI) adjusting for age, passive smoke exposure, smoking status, saturated fat intake and vegetable intake. Concentrations >121 Bq m^{-3} were associated with a >3-fold increase in the interaction OR (OR= 3.26; 95% CI = 1.1, 10.1) for *GSTM1* null cases compared with *GSTM1* present cases. An exposure-response trend was also evident (p trend = 0.04). This is the first study to provide evidence of a Rn-222 and *GSTM1* interaction in risk of lung cancer. However, the sample size was small and further research is warrant.